white streaks. The streaks are the scars of the paths taken by the larvae as they migrate through the liver. The larvae finally come to rest as little glistening beads of jellylike material on the membranes supporting the intestine.

Each such little bead is an embryonic tapeworm, which must now await consumption by a carnivorous animal, such as a dog. In its intestine the embryo will grow into a mature tapeworm; its eggs later escape in the dog's droppings and can infect another rabbit.

Disfigured livers are objectionable and often prejudice the value of the entire carcass. To prevent this type of infection in rabbits, the owner has to keep all feed, water, nest-box straw, equipment, and everything the rabbit may touch entirely free of possible contamination with droppings of carnivores, particularly dogs. He should never permit a dog to sleep on feed sacks and bales of hay or straw or to contaminate anything that will reach the rabbits. And, conversely, to keep the dog from becoming infected, he should not allow it to feed on the viscera of infected rabbits.

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Diseases and Parasites of Minks

JOHN R. GORHAM

THE BOTULISM bacterium, Clostridium botulinum, produces a potent toxin in contaminated meat. Such meat, fed to minks, can kill them in 18 to 96 hours. Almost immediately the animals suffer muscular incoordination and stiffness. Paralysis of the front or hind legs follows. The muscles used in breathing become paralyzed by the toxin, and the minks die.

The carcasses of sick horses and spoiled feed that has had a chance to become warm for a time should never be used.

The treatment of minks affected with botulism often is to no avail. Prompt recognition by the rancher will give the veterinarian a better chance to save the minks that have not eaten enough of the poisoned food to show symptoms. The owner should throw away immediately all the contaminated, frozen, stored food and the uneaten ration in the pens.

Antitoxin that contains toxin-neutralizing antibodies against the three common types of toxins, designated A, B, and C, should be administered to every mink on the ranch as soon as possible.

For prevention, a toxoid—preventive inoculation—offers fair promise of being helpful. The first work on botulism toxoids for minks was done in Sweden. Following the disastrous outbreaks of botulism in this country when spoiled whale meat was fed, research workers in Utah and Wisconsin and at the Department of Agriculture Fur Animal Disease Station developed an experimental toxoid, which is available commercially. It is emphasized, however, that care in management and selection of feed is
the best insurance for preventing this malady.

**Anthrax** is a fatal disease of minks caused by the bacterium *Bacillus anthracis*. Minks die within a few days after eating meat from an anthrax-infected cow, pig, or horse. The animals show few if any signs of sickness before death.

An autopsy discloses that the spleen is enlarged, blue black, and easily torn. Because other disease conditions in minks seem to be similar to anthrax, a diagnosis can only be made on bacteriological examination in a laboratory. The surest prevention is to avoid feeding meat from sick animals.

Penicillin injections have been given to affected minks with good results.

People may become infected by handling pelts removed from minks that died from anthrax. Because the spores—the resistant stage of the bacterium—survive the tanning process, dead animals should be burned and not pelted. Premises once contaminated remain infected.

**Abscesses** caused by Micrococcus and Streptococcus and by *Klebsiella ozaenae* are common in minks. Often slivers of bone or barbed grasses injure the animal and carry the bacteria into the skin or lining of the mouth. Pus accumulates, and an abscess, or boil, is formed.

When the abscess feels hot and swollen and is fluctuating to the touch, one should make a vertical incision long enough to drain away the pus with a sharp, sterile blade. The cavity should be washed out with a mild disinfectant, such as dilute hydrogen peroxide, and dusted with sulfa powder, such as sulfathiazole or sulfanilamide. A veterinarian can demonstrate this simple technique. In all cases, an injection of 100,000 to 200,000 units of penicillin should be given.

**Streptococcus** organisms—the small bacteria which, when viewed under the microscope, occur in chains like a string of beads—may cause other troubles. Ranchers may notice that the top and sides of the head, nose, and upper neck region of some of their minks have suddenly become swollen. This condition is called cephalic cellulitis. The minks are sluggish and refuse food. If they are not given an inoculation of penicillin at this time, death will ensue.

These types of bacteria may also cause fatal septicemia (blood poisoning) in young minks. The symptoms are not apparent, and the animals are found dead in their nest boxes. Penicillin is the antibiotic most frequently recommended for treating streptococcal infections.

*Pseudomonas aeruginosa* causes a rare but deadly disease in minks. This bacterium is introduced into the herd through contaminated drinking water. Most minks are sick only an hour or two before they die—they may look healthy in the evening but are found dead the next morning. Pneumonia is the most significant finding at autopsy. Streptomycin and polymyxin are the antibiotics of choice. As these drugs are highly toxic to minks, information as to the dosage and manner of treatment should be obtained from a veterinarian.

*Pasteurella multocida* is found in cases of mink pneumonia. The disease often appears in the spring and fall when the weather is damp and the temperature is subject to rapid change. The nest boxes should be constructed so that they can be kept dry and well ventilated. Minks can stand freezing temperatures, but they cannot tolerate wet nest hay and boxes.

Rapid, shallow breathing, as evidenced by a heaving of the flanks, is the most important clinical sign of the malady.

For treatment, injections of penicillin, Aureomycin, or Terramycin can be given. Treatment is not always successful because the animal is often too far gone by the time it looks ill.
Gangrene occasionally develops in wounds and after contaminated tissue vaccines have been injected. Various bacteria have been isolated—Clostridium, Streptococcus, Micrococcus, and Actinomyces.

Animals that have been given contaminated vaccine drag the inoculated leg. Gas bubbles form in the muscle and a foul discharge is noted later.

The broad-spectrum antibiotics, such as Aureomycin and Terramycin, are used for treatment.

Pleuritis, or inflammation of the lining membranes of the chest cavity, often causes death. When the ribs are cut and the chest cavity is opened, a large amount of thick, grayish-yellow pus is seen. The lungs are compressed and covered with adhesions.

Many bacteria have been isolated from this pus, including Streptococcus and Micrococcus species, coliforms, Norcardia species, Actinomyces necrophorus, and organisms like pleuropneumonia.

Animals sick with pleuritis are rarely treated, because they usually are found dead with no signs of illness.

Enteritis, an inflammatory condition of the small and large intestine, is commonly observed on mink ranches in the United States and Canada.

Spoiled food, corrosive chemicals, and a specific virus can cause it. Many bacteria have been suspected, but the actual cause in many instances is unknown. The disease is more apt to develop in the summer months.

Affected minks often have a big appetite. Beneath their pen is seen a large accumulation of black, tarry, or grayish-white droppings. Usually only a few minks on the ranch are affected.

When the whole herd is involved, prompt veterinary assistance is required, because sulfa drugs or high levels of antibiotics will have to be added to the rations.

When distemper is mentioned, mink ranchers everywhere have cause for alarm, for it is very serious in minks throughout the United States.

Although almost all of the minks get the disease in any given outbreak, some animals do not show any symptoms. The number of deaths may vary from one outbreak to another. The loss may be as high as 90 percent in young mink, but the average loss in older mink is 30 to 40 percent. Kittens as young as 3 weeks may be infected. Outbreaks can occur at any time.

The incubation period—the interval between the time the virus first enters the mink and the appearance of disease—is about 9 to 14 days.

The first sign of the disease is swollen, watery eyelids. The lids become crusty and stick together in 2 or 3 days. The minks may have an excellent appetite at this time.

Next the feet may swell—the so-called "snowshoe" foot. Small, brownish, granular scabs can be seen on the surfaces of the pads. The minks may die at this time or appear to recover, but these "recovered" individuals may later die of what the rancher calls the "screaming fits." In this type of distemper, the virus has invaded the brain, causing the minks to froth at the mouth, chew violently on the wire netting, roll about the pen, and scream sharply. These convulsive seizures may last a short or a long time. The mink usually dies after one or two attacks.

The virus is spread from animal to animal through the air. In the normal breathing process, minks that show distemper (as well as the apparently healthy carriers) emit millions of small, virus-containing droplets into the air. The virus also is found in the saliva, nasal secretions, and skin scurf shortly before and during an attack. A mink may also shed virus into the air after the signs disappear.

Distemper can be transmitted among mink by indirect means, such as contaminated mitts and food pans.

A diagnosis often can be made by observing affected animals, but sometimes laboratory tests are necessary.
Microscopic slides can be made of tissue from the bladder and trachea (windpipe) and stained with special dyes. Distemper leaves rather characteristic marks, called inclusion bodies, in those tissues. They are small stained "dots," or specks, which represent distemper virus.

The surest way to obtain a diagnosis is to remove the spleen from the suspected distemper-infected mink, grind it in a liquid suspension, and inject it into a ferret. If the spleen from the animal in question contains active virus, the ferret, which is almost 100 percent susceptible to the virus, will become infected.

Every precaution must be made to keep distemper away from the ranch. New breeding stock or minks returned from fur shows should be quarantined for 2 months, as they may carry the virus into the herd.

Infected dogs are a dangerous source of distemper virus for minks.

Two general types of distemper vaccine are on the market—the killed-virus vaccines and the living modified-virus vaccines.

The killed-virus vaccines, prepared from a spleen containing virulent virus of an animal dead of the disease and treated with formalin to inactivate the virus, have some disadvantages. Animals inoculated with them develop immunity rather slowly. The immunity seems to leave the animal, and so revaccination is necessary.

Living-virus vaccines, which are made by growing the virus in chicken embryos, have been more successful in controlling outbreaks.

Virus enteritis has been considered a serious threat to minks in the United States. It has been found in Ontario and has been reported on a few ranches in Wisconsin and Illinois. The disease may kill as high as 80 percent of the kitten crop. It spreads rapidly on a ranch.

The first indication of sickness, within a week after the minks are exposed, is a sudden loss of appetite. If the feces are examined, a slightly pink to grayish-white "slug" (intestinal cast) can be noticed. The animals that do not die within 4 or 5 days recover, but may never reach maximum size. When the dead animals are opened, the intestines appear bright red.

Minks that have recovered from the disease should be pelted in season, because they may harbor the disease as "silent" carriers and serve as a source of virus for the kitten crop of the following summer.

Because virus enteritis might be related to feline panleukopenia virus, cats and visitors and other traffic should be excluded from the mink yards. Research workers have tried to develop a vaccine.

Good sanitary practices are the only control measures.

Nursing sickness, a common condition of nursing minks, appears before the fifth or sixth week of lactation. Affected females are first noticed in late June at about the time the young minks are weaned, but the disease may appear after the kits have been taken away.

Symptoms of nursing sickness include lack of appetite, loss of flesh, weakness, and incoordination. Death follows a period of coma. Some females, although apparently starving, fill their mouths with feed but do not swallow it.

The carcass is extremely thin—there is almost no body fat. A yellow, easily torn liver is often observed. Usually the gall bladder is distended with bile—evidence the animal has not been eating—and the stomach is empty.

Ranchers have considered the prevention of this condition a management problem. The nursing females should be carefully watched. If any become thin and dehydrated, the kittens should be given to another female if they are too small to be weaned. Another help is to make food and water easily available to the young kits from an early age onward.
Dr. G. R. Hartsough, of the Great Lakes Ranch Service, has suggested that the cause of nursing sickness may be a depletion of salt in the diet. He has recommended the addition of 0.5 percent of table salt to the feed from late May until the middle of July. Because some commercial mink cereals contain salt, caution should be used when adding more salt, as minks are susceptible to salt poisoning.

Urinary calculi, called "gravel" or "stones" by the rancher, causes heavy losses. The disease is observed rarely in the Pacific Northwest, but calculi are a major problem among the mink herds of Wisconsin and other North Central States.

Most deaths occur in pregnant, whelping, or lactating females. Losses of females on some ranches have exceeded 15 percent.

Bladder stones often hinder the passage of young through the birth canal. After prolonged labor, the female becomes weak and dies within a day or two. Sometimes ranchers report straddling gait, difficult urination, and paralysis. When stones are found at autopsy and have caused death of the mink, the calculi are frequently accompanied by a discharge of blood and pus.

In male minks, small calculi can lodge at the penis bone and block the flow of urine to the outside.

The cause is unknown. The stones are almost pure magnesium ammonium carbonate. Because a calculus of this type is thought to form in alkaline urine, investigators believe this alkalinity has a role in the disease. Experiments have been started in an attempt to find out whether the bacteria in the bladder or the ration itself cause an alkaline urine.

Observations have indicated that the feeding of ammonium chloride from the first of April through June may help control the disease, because the drug helps to establish a more neutral urine. It is important that the animals be given ample water.

Steatitis (yellow fat) has been recognized as a disease since 1942. The malady undoubtedly occurred before that time but was diagnosed as something else.

It affects only young minks and occurs in the summer and fall. The disease gives no warning—a fat kitten, apparently in the best of health, refuses the night feeding and is dead in the morning. Before the cause was known, it forced many ranchers to go out of business.

Young minks succumbed when they were fed rations containing high percentages of fish scrap that had been in storage for a long time. Storage horsemeat that has quantities of untrimmed fat also caused the disease. Observations of field outbreaks and laboratory experiments have revealed that these diets have a high content of unsaturated fats and are deficient in vitamin E. Apparently those two factors work together to form the characteristic yellow-brown color of the fat and eventual death.

Often the ailment occurs soon after weaning; therefore the dam, as well as her kits, should have a diet that contains ample vitamin E. This vitamin may be obtained in many different forms (stabilized powder, wheat germ meal, wheat germ oil, and purified tocopherols).

It has been our experience that the stabilized powder is the most satisfactory.

It is possible that the liquid preparations are more readily oxidized by the rest of the ration, so that some of the vitamin E is withdrawn. Vitamin E is a dietary essential that the body is unable to synthesize (manufacture) and must be obtained from outside sources. It is also called the antisterility factor and tocopherols. Vitamin E protects other vitamins from the destructive effects of oxygen and is thought to be necessary for normal reproduction in some animal species.

Ranchers should supplement the diet of the young growing kits so that the amount of wheat germ meal or
synthetic preparations in the ration provide 15 milligrams of vitamin E per day per mink.

In an outbreak, the owner should lower the level of fish or storage horsemeat, substitute fresh, unfrozen horsemeat and liver, and add wheat germ meal or stabilized vitamin E powder to the diet. Each affected kit should receive an inoculation of 15-20 milligrams of vitamin E a day for 3 days or more. The preparations can be obtained at drugstores or from veterinarians. They are sold under such names as injectable tocopherols.

**HYDROCEPHALUS**, or "water head," is a hereditary disease that causes death in minks.

The distended head, malaise, incoordination, and lack of growth are readily seen when the litters are first checked after whelping.

The accumulation of fluid within the cavities of the brain forces the brain tissue against the soft developing skull bones and pushes them outward. Little brain tissue is found when the young mink is examined after death.

Because the affected kitten usually dies before it is 6 weeks old, it should be removed from the litter, as it will take nourishment from the dam.

The dam, sire, and littermates of the hydrocephalic kit should be pelted the following winter. Both parents are sure to carry the recessive genes that are responsible for the disease, and all or most of the littermates may carry it. This manner of control, if followed annually, should eventually eliminate the malady from the ranch.

**THE CAUSE OF WET BELLY** in male minks is unknown. The constant dribbling of urine causes a loss of hair and a severe inflammation of the skin and underlying tissues in the region of the penis. Frequently 10 percent of the males on a ranch may be affected.

When the animal is pelted and the skin stretched on the board to dry, the damaged area is readily visible. Because those pelts bring lower prices at auction, the condition is a cause for concern.

The addition of molasses and fresh liver to the ration has been recommended.

**SPORADIC AILMENTS** may plague mink ranchers. Fatty or yellow liver is often found in a postmortem examination. It should not be considered a separate disease, but rather an abnormal finding, which accompanies such diverse conditions as nursing sickness, food poisoning, poisoning by chemicals, pregnancy disease, and uremia.

Minks often are addicted to fur chewing. They have a lionlike appearance, because they chew off all hair they can reach. Sometimes a mink will chew off its entire tail—hair, flesh, and bone—and continue chewing until it kills itself.

Hot, humid, still days can cause the death of old and young minks alike. If the female is whelping or if the kits are only a week or two old, the losses are high. The female refuses to nurse the kits, or she may become so restless that the young minks are dragged about until dead.

Mink farmers should never feed chicken byproducts without making sure that the chicken heads do not contain diethylstilbestrol (caponizing) pellets. Diethylstilbestrol, even in small amounts, will cause breeding failures in male and female minks.

(Diethylstilbestrol is a synthetic hormone-like substance that depresses the development of male characteristics and, in females, interferes with the normal reproductive cycle. It has been used to bring about increased weight gain in steers and feed-lot heifers and to stimulate the rate of gain in fattening lambs. It is frequently used in turkeys and chickens to reduce their activity and to increase weight gains.)

A condition causing death in minks that carry the Aleutian gene—that is, Aleutian and Sapphire color phase mutations—results in occasional losses. The affected individual may exhibit
bleeding at the mouth, become extremely thin, and succumb in a month or less. To increase the hardiness of this beautiful mutation, many ranchers outcross them with standard black minks. No effective treatment is yet known.

The gray fleshfly (of Wohljahriia species) is responsible for the death of many young minks. The female fly deposits eggs, which are quickly transformed into moving maggots on the skin of the young kitten. The maggots bore into the skin and leave a deep sore behind them.

The young minks become extremely restless and cry pitifully. Infection, lack of appetite, and exhaustion frequently lead to death.

The kits that have maggots should be removed and treated by injecting hydrogen peroxide, chloroform, or ether into the opening; these substances usually make the maggot back out. The wound should then be treated with an antiseptic, such as mild tincture of iodine.

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**Diseases and Parasites of Foxes**

**JOHN R. GORHAM**

THE IMPORTANCE of cleanliness in the care of foxes cannot be over-emphasized. With them, as with many other animals, it is easier to prevent a disease than to cure it.

Foxes are hearty, vigorous animals and rarely are sick if they have an adequate diet and healthy conditions in which to live. They must be reared in pens with woven-wire bottoms. That method of husbandry disrupts the life cycle of many disease agents and parasites, because the feed does not become tainted with droppings.

To make the use of disinfectants easier, the equipment and construction should be designed to save labor. Simple, handy pens, kennels, feed houses, and feeding equipment are best. Because fecal matter and other organic material harbor bacteria and viruses, cleaning should precede disinfection. One agent can do both jobs.

When routinely cleaning and disinfecting pens and kennels, ordinary lye solution is an effective and economical disinfectant. One can of lye, which contains 13 ounces, is enough to make 15 gallons. If large areas must be covered, the lye can be bought as caustic soda and each pound will make about 20 gallons of solution. The solution does not have to be heated.

Besides acting as a disinfectant, lye solutions cut grease and partly dissolve and penetrate fecal material, but lye has some disadvantages.

Concentrated lye is a poison. It is destructive to aluminum, paints, and clothing. It does not harm wood or iron in the dilute concentration recommended. The dilute solution as recommended is not harmful to the animals in the amounts which might remain in the pens. Quantities of the solution should not be left unguarded where the animals might drink it. The solution may be slightly irritating to the hands and face of the operator. Since exposure to the air soon converts lye